PRIVATE AND SOCIAL COSTS OF GROWING CULTIVARS SUSCEPTIBLE TO DISEASES

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Agricultural Research Institute, Wagga Wagga NSW 2650

ABSTRACT

With recent deregulation, crop cultivars without adequate resistance to the major diseases can be released from breeding programs and grown by farmers. Farmers who grow susceptible cultivars impose costs on other farmers, since the inoculum of some diseases can spread over long distances. Growing susceptible cultivars increases the amount of inoculum that reaches resistant crops, thus placing the resistance of those crops at risk. Because diseases occur episodically, the private costs of a susceptible cultivar can be perceived as low in situations where the social costs of such cultivars can be important. In this paper, analysis is presented to show that the social costs can indeed be significant in relation to the private costs. The policy options in the face of these social costs are explored, to determine if there is a valid role for government intervention where such market failure is evident. The difficulties of finding an appropriate government response in a deregulated industry highlight some of the often-overlooked costs of deregulation.

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1. INTRODUCTION

Farmers aiming to maximise their utility function face many issues in their cultivar selection. They have only a limited number of choices, at least partly because cereal cultivars available to farmers in Australia have been regulated. State authorities have reserved the right to release or not release new cultivars for farmers. In addition, the Australian Wheat Board (AWB) has traditionally (before its monopoly powers on the domestic market were removed in 1988) imposed "dockages" or specific payment discounts on wheat cultivars they have designated as not suitable quality.

When cultivars have been released from breeding programs with the approval of the authorities, they generally have had some resistance to the diseases that were considered economically important in that particular environment. However, with time, new pathotypes may arise and build up, such that the resistance is no longer effective or "breaks down". If the disease is significant, then the losses from continuing to grow that cultivar are expected to be sufficiently high as to outweigh any other advantages of that cultivar, and a replacement cultivar is required. As a result, there has been a continuing relatively rapid turnover of cultivars used in the wheat industry (Brennan 1989).

For diseases where chemicals have been the major control measure, the risk of development of tolerance of the pathogen to the fungicide is analogous to the development of new pathotypes (Staub 1991).

Until the deregulation of the domestic market for wheat in Australia in the late 1980s, authorities had control over the cultivars released. Since deregulation, any cultivar may be released to growers. Some growers, faced with increased returns from particular types of wheat (for example, feed wheats for beef feedlots), are now considering the use of susceptible cultivars. It is therefore appropriate to consider the issue of private and social costs of growing susceptible cultivars. While there have been some estimates of the expected (private) losses to farmers who continue to grow susceptible cultivars (eg, Brennan and Murray 1989), there has not been an analysis of the social costs of growing susceptible cultivars.

In this paper, the private costs of wheat diseases are examined in section 2. Issues relating to public risk diseases giving rise to social costs are examined in section 3, where the social costs of susceptible cultivars are defined and discussed. In section 4, an empirical evaluation is made of the likely importance of social costs. Possible policy implications are discussed in section 6, and some conclusions are drawn in the final section.
2. PRIVATE COSTS OF SUSCEPTIBLE CULTIVARS

The private costs of wheat diseases in Australia have been estimated at $401 million per year (Table 1). The potential costs of diseases if there were no controls used at present would be considerably higher. It is not meaningful to total the potential costs from each disease, since if one major disease reached its full potential, the additional potential losses from extra diseases would be reduced (Brennan and Murray 1989).

The figures in Table 1 are based on the estimated losses that would be incurred by farmers, so that the total private costs of growing susceptible cultivars could be taken as gains from resistance plus the present losses for diseases that have some potential control available from genetic resistance. Brennan and Murray (1989) estimated that resistance contributed over $50 million per year for each of major foliar diseases, and more than $14 million for a further three diseases. Therefore the private costs of growers using susceptible wheat cultivars in Australia would be markedly higher than the current estimated losses.

3. SOCIAL COSTS OF SUSCEPTIBLE CULTIVARS

3.1 Public Risk Diseases

3.1.1 Concept of public risk diseases

Plant diseases can readily spread from one crop to a neighbouring one and, in some instances, to more distant crops. A public risk disease can be defined as one that is a threat to the productivity of crops other than that in which the disease first occurs. Some diseases constitute a greater public risk than others, but there has been only limited literature on the concept. By way of contrast, many authors have referred to the hazards of genetic uniformity (Van der Plank 1968, National Academy of Sciences 1972, Marshall 1977). There has also been discussion on the public risk of genetically engineered organisms (Gillett 1986, Gould 1988) and the threat of exotic diseases (Kingsolver et al. 1983, Pemberton 1988, Yang et al. 1991).

3.1.2 Categories of public and private risk diseases

Some diseases constitute a greater public risk than others. There are various categories of public and private risk according to features of the individual diseases. These include:

(a) Means of dispersal - long or short distance for primary and/or secondary dispersal;
(b) Pathogen variation status - whether any variation of agricultural significance has been reported;
(c) Number of cycles within the growing season - monocyclic or polycyclic.
<table>
<thead>
<tr>
<th>Disease</th>
<th>Cost per ha($)</th>
<th>Total costs ($m)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Potential</td>
<td>Present</td>
</tr>
<tr>
<td>Diseases of Foliage</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stem rust</td>
<td>10.85</td>
<td>0.38</td>
</tr>
<tr>
<td>Stripe rust</td>
<td>14.28</td>
<td>1.85</td>
</tr>
<tr>
<td>Flag smut</td>
<td>4.56</td>
<td>0.02</td>
</tr>
<tr>
<td>Yellow spot</td>
<td>5.12</td>
<td>2.11</td>
</tr>
<tr>
<td>Septoria tritici blotch</td>
<td>12.87</td>
<td>5.00</td>
</tr>
<tr>
<td>Leaf rust</td>
<td>2.59</td>
<td>0.37</td>
</tr>
<tr>
<td>Septoria nodorum blotch</td>
<td>5.35</td>
<td>5.32</td>
</tr>
<tr>
<td>Yellow dwarf</td>
<td>0.26</td>
<td>0.25</td>
</tr>
<tr>
<td>Powdery mildew</td>
<td>0.35</td>
<td>0.30</td>
</tr>
<tr>
<td>Septoria avenae blotch</td>
<td>0.02</td>
<td>0.02</td>
</tr>
<tr>
<td>Bipoaeros leaf spot</td>
<td>0.02</td>
<td>0.01</td>
</tr>
<tr>
<td>Downy mildew</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>Milo spot (Ring spot)</td>
<td>0.06</td>
<td>0.06</td>
</tr>
<tr>
<td>- Sub-total</td>
<td><strong>15.70</strong></td>
<td><strong>185</strong></td>
</tr>
<tr>
<td>Diseases of Root and Stem Base</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Take all</td>
<td>14.88</td>
<td>6.89</td>
</tr>
<tr>
<td>Crown rot</td>
<td>1.65</td>
<td>1.11</td>
</tr>
<tr>
<td>Rhizoctonia bare patch</td>
<td>3.64</td>
<td>0.50</td>
</tr>
<tr>
<td>Common root rot</td>
<td>2.81</td>
<td>1.87</td>
</tr>
<tr>
<td>Cereal cyst nematode</td>
<td>10.53</td>
<td>4.58</td>
</tr>
<tr>
<td>Root lesion nematode</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- (P. thornei)</td>
<td>1.16</td>
<td>0.33</td>
</tr>
<tr>
<td>- (P. neglectus)</td>
<td>0.03</td>
<td>0.03</td>
</tr>
<tr>
<td>Eyespot</td>
<td>0.82</td>
<td>0.19</td>
</tr>
<tr>
<td>Pythium root rot</td>
<td>0.04</td>
<td>0.03</td>
</tr>
<tr>
<td>Pyrenophora seed rot</td>
<td>0.01</td>
<td>0.01</td>
</tr>
<tr>
<td>- Sub-total</td>
<td><strong>15.53</strong></td>
<td><strong>183</strong></td>
</tr>
<tr>
<td>Diseases of Inflorescence and Kernel</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bunt</td>
<td>30.68</td>
<td>0.00</td>
</tr>
<tr>
<td>Loose smut</td>
<td>1.88</td>
<td>0.16</td>
</tr>
<tr>
<td>Black point</td>
<td>4.45</td>
<td>2.60</td>
</tr>
<tr>
<td>Ergot</td>
<td>1.61</td>
<td>0.00</td>
</tr>
<tr>
<td>Pink grain (Scab)</td>
<td>0.93</td>
<td>0.00</td>
</tr>
<tr>
<td>- Sub-total</td>
<td><strong>2.76</strong></td>
<td><strong>33</strong></td>
</tr>
<tr>
<td>Total</td>
<td><strong>33.99</strong></td>
<td><strong>401</strong></td>
</tr>
</tbody>
</table>

To give an illustration, the main diseases of wheat are classified according to these categories in Table 2. Those classified as L-V-P pose the greatest public risk, while the S-N-M diseases pose the least public risk.

For example, the rusts, with their history of variation, polycyclic nature and ability to be transmitted long distances (both for primary and secondary inoculum), are in the highest risk category. By way of contrast, most root diseases have limited secondary distance dispersal, no pathogenic variation and are monocyclic, so pose little or no risk to adjacent crops.

In the intermediate categories are diseases whose secondary inoculum is transmitted short distances and are monocyclic but have a history of new pathotypes (e.g., loose smut); and diseases transmitted long distances for primary dispersal and short distances for secondary dispersal (e.g., Septoria tritici blotch).

Table 2: Categories of Public Risk for Wheat Diseases

<table>
<thead>
<tr>
<th>Disease</th>
<th>Distanceᵃ</th>
<th>Variationᵃ</th>
<th>Cyclingᵃ</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>High public risk</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stem rust</td>
<td>L</td>
<td>V</td>
<td>P</td>
</tr>
<tr>
<td>Leaf rust</td>
<td>L</td>
<td>V</td>
<td>P</td>
</tr>
<tr>
<td>Stripe rust</td>
<td>L</td>
<td>V</td>
<td>P</td>
</tr>
<tr>
<td>Barley yellow dwarf</td>
<td>L</td>
<td>V</td>
<td>P</td>
</tr>
<tr>
<td><strong>Intermediate public risk</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Septoria tritici blotch</td>
<td>L</td>
<td>V?</td>
<td>P</td>
</tr>
<tr>
<td>Septoria nodorum blotch</td>
<td>L</td>
<td>V?</td>
<td>P</td>
</tr>
<tr>
<td>Yellow spot</td>
<td>S?</td>
<td>N</td>
<td>P</td>
</tr>
<tr>
<td>Flag smut</td>
<td>S</td>
<td>N</td>
<td>M</td>
</tr>
<tr>
<td>Loose smut</td>
<td>S</td>
<td>V</td>
<td>M</td>
</tr>
<tr>
<td><strong>Low public risk</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eyespot</td>
<td>S</td>
<td>?</td>
<td>P/M</td>
</tr>
<tr>
<td>Cereal cyst nematode</td>
<td>S</td>
<td>N</td>
<td>M</td>
</tr>
<tr>
<td>Root lesion nematodes</td>
<td>S</td>
<td>N?</td>
<td>M</td>
</tr>
<tr>
<td>Take all</td>
<td>S</td>
<td>N</td>
<td>M</td>
</tr>
<tr>
<td>Common root rot</td>
<td>S</td>
<td>N</td>
<td>M</td>
</tr>
<tr>
<td>Crown rot</td>
<td>S</td>
<td>N</td>
<td>M</td>
</tr>
</tbody>
</table>

ᵃ: Distance dispersal L long, S short; V variation, N no variation; Cycling M monocyclic, P polycyclic; ? uncertain (data not available)
3.2 Social Costs of Susceptible Cultivars

The focus of this paper is the high and intermediate categories of public risk, where there is a greater chance of incurring a social cost. Growers who use cultivars that are susceptible to diseases with high public risk not only place their own production at risk (that is, have a "private" cost), but also increase the likelihood of the pathogen producing a new variant able to overcome other resistances (Johnston et al. 1983). Thus, farmers growing susceptible cultivars are imposing a cost on their neighbours by increasing the susceptibility of their neighbours' crops to disease losses. The magnitude of the external cost will vary with the disease, the cultivar grown and factors such as the total area grown and the proximity of other crops.

This "social" or "external" cost imposed by growing susceptible cultivars has not been subjected to economic evaluation to assess the case for regulation or restrictions on the cultivars that can be released or grown. The issue is particularly important in today's deregulated industry, where susceptible cultivars can be released without official sanction.

There are three different forms of externality or social cost resulting from susceptible cultivars. First, there is a direct yield loss for (generally neighbouring) crops from spore dispersal from the susceptible crop. Second, there is an indirect cost to other growers through the loss of breeding materials and interrupted flow of cultivars when mutation to virulence occurs in currently-used cultivars. Third, there are additional indirect costs from "bridging" whereby the use of a susceptible cultivar provides the pathogen with the means of accumulating the necessary virulences to attack other cultivars with more complex resistances. For example, the cultivar Cook with the stem rust resistance genes $Sr5$, $Sr6$ $Sr8a$ and $Sr36$ undoubtedly had its effective life shortened by the widespread use in central Queensland of the cultivar Oxley with $Sr5$, $Sr8a$ and $Sr12$ during the early 1980s. Had Oxley not been grown, it is likely that the pathogen would have had to accumulate two virulence genes in order to become virulent on Cook. Because Oxley continued in production after the advent of the strain virulent on Oxley, only a one step change was necessary. Mutation is the most likely means of gaining the additional virulences in this instance (Luig 1983) and the probability of a two gene mutation are much less than for a one gene mutation.

3.3 Factors Affecting Importance of Public Risk

Fungal diseases such as the rusts develop when three criteria are met (Watson 1974):
   (a) the fungus is present,
   (b) climatic conditions are suitable, and
   (c) susceptible cultivars are grown.

These criteria may all be quantified for certain wheat diseases. For example, surveys can give some measure of the availability of the inoculum. The relationship between
weather conditions and various crop disease epidemics has been identified and modelled for a number of diseases including the wheat rusts (Teng and Bowen 1985, Roelfs 1985). Finally, some measure of the areas sown to susceptible and resistant cultivars can be obtained.

Susceptible crops produce inoculum of the disease, thereby increasing the risk of mutation occurring with respect to the host genes currently protecting resistant crops. Susceptible crops also provide a host for the establishment of rusts of foreign origin which have entered Australia on rare occasions (Watson 1974, Luig 1985) and genetic recombination between different pathogen genotypes (Watson 1981).

A key issue relating to likelihood of susceptible crops leading to pathogenic mutation is the mutation rate to virulence (that is, the rate at which the pathogen mutation occurs to become virulent to the host). These mutation rates vary widely for different genes (Kiyosawa 1977, 1982, Luig 1979, Schafer and Roelfs 1985). For example, mutations to virulence for Sr26, the most durable gene for stem rust, have never been found (Luig 1985), even though cultivars with resistance based on that gene have occupied large areas over more than twenty years in Australia. There have been estimates made of relative mutation rates for a number of stem rust resistance genes (Luig 1983, Schafer and Roelfs 1985).

### 3.4 Economic Issues with Public Risk Diseases

There are a number of policy options in response to the social costs of public risk diseases. Analysis is required to establish the most desirable form of regulations or taxes, their magnitude and whether the policy costs of imposing them (information, administration and enforcement costs) would be outweighed by the benefits. For example, the maintenance of a varietal dockage scheme is more difficult in the current deregulated market than it would have been with complete regulation. The added difficulties and their cost need evaluation.

In addition to the external costs of susceptible cultivars discussed above, there are other aspects of the use of susceptible cultivars that need to be considered: (a) costs to farmers from reduced plantings of susceptible cultivars, and (b) policy costs.

The cost to farmers of reducing their areas of susceptible cultivars is conceptually as well as practically difficult to assess (Johnston et al. 1983). Expected yield and general agronomic performance will obviously be factors of importance to farmers in their choice of cultivars in the absence of dockages for disease susceptibility but so will factors such as their optimum sowing time, grazing value and the farmers' knowledge. In some cases, significant areas of susceptible cultivars have continued to be grown even though resistant cultivars were available which produce yields just as high or higher than susceptible cultivars. Information costs or some other personal factors presumably explain this choice by farmers. It would be most difficult to assess what losses in total utility farmers would suffer from reductions in the area of these susceptible crops (Johnston et al. 1983).
Most susceptible cultivars in the wheat industry result from the "breakdown" of resistant cultivars rather than the breeding or release of susceptible ones. However, cultivars exhibit degrees of resistance rather than being simply susceptible or resistant and breeding organisations must consider whether they will release cultivars with low degrees of resistance.

There is little incentive for farmers to grow high yielding but disease susceptible cultivars because the farmer stands to bear a private cost from disease loss, in addition to the external cost imposed on neighbours. In these circumstances, the quantities of disease susceptible wheat grown are not likely to be large. Nevertheless, since deregulation, the issue has become an important one and needs further economic investigation.

4. EMPIRICAL ANALYSIS

4.1 Analytical Framework for Assessing Impact of Susceptible Cultivars

4.1.1 Analyses of pest/disease control

There is no established framework for the economic analysis of the cost of disease susceptibility or alternatively the value of crop disease resistance. One form of analytical model used for pest control decisions is the treatment of pests as a common property resource (for example, Regev, Gutierrez and Feder 1976, Briggs 1989), although that model has been used for insect pests rather than crop diseases. These studies distinguished between the gains made by each decision-maker (farmer) and those made by society. Different functions were used for each, and then the optimal control levels compared for each, and if a significant difference emerged from the two viewpoints, then it was apparent that a non-regulated market would not yield the optimal solution from society's point of view.

The pest control model can be classified into three component parts (Briggs 1989): (a) crop damage abatement; (b) pest ecology; and (c) crop production. The crop damage abatement component of the model relates to the efficacy of disease resistance in reducing crop damage (either yield loss or quality impairment). Disease resistance does not increase potential output (it may reduce it if there are genetic linkages to yield penalties), but may increase realised yields by reducing damage in the presence of the disease (Lichtenberg and Zilberman 1986).

A specification used to describe crop damage abatement used in several studies (e.g. Talpaz and Borosh 1974, Moffitt and Farnsworth 1981), has the cumulative density function given by:

\[ A = 1 - \exp(-aX^b), \quad X \geq 0, \quad b \geq 0 \]

where \( X \) is "dose" of control measure (resistance in this case) and \( A \) represents the degree of protection offered by disease resistance such that \( 0 < A < 1 \). A version of
the special case where $b=1$.

This model may be useful for the evaluation of optimal strategies to combat disease losses. However, it is not an ideal tool for identifying the social cost of susceptible cultivars. For this paper, an alternative form of analysis was sought.

4.1.2 General model of probability of resistance "breakdown"

An alternative analytical framework is to use probability theory, treating the "breakdown" of resistance in a cultivar as a random event occurring with an identifiable probability. No previous work using probabilities to address this question has been found.

Assume for simplicity that two cultivars (cultivars 1 and 2) are grown in a given region in a given year. Let the level of resistance of a cultivar be represented by a value between 0 (immune, highly resistant) and 1 (highly susceptible). The probability of "breakdown" of resistance in cultivar 1 in year $t$, $B_{1t} (0 \leq B_{1t} \leq 1)$, is a function of the current level of resistance in that cultivar ($R_{1t}$) and the expected amount of inoculum available to that cultivar in that year ($I_{1t}$).

$$B_{1t} = f(R_{1t}, I_{1t}).$$

The expected amount of inoculum, in turn, is a function of the current level of resistance ($R_{1t}$), the area sown to cultivar 1 in year $t$ ($A_{1t}$), the amount of inoculum from cultivar 2 ($I_{2t}$), and the weather conditions affecting the disease ($W_t$):

$$I_{1t} = f(R_{1t}, A_{1t}, I_{2t}, W_t).$$

The expected amount of inoculum from cultivar 2 is a function of the level of resistance in that cultivar ($R_{2t}$), the area sown to that cultivar ($A_{2t}$), the mean distance of crops of cultivar 2 from cultivar 1 ($D_t$), and the weather conditions ($W_t$):

$$I_{2t} = f(R_{2t}, A_{2t}, D_t, W_t).$$

Thus, combining equations (2) to (4), we get

$$B_{1t} = f(R_{1t}, A_{1t}, R_{2t}, A_{2t}, D_t, W_t),$$

where

- $R_{1t}$ is the level of resistance of cultivar 1 in year $t$,
- $A_{1t}$ is the area sown to cultivar 1 in year $t$,
- $D_t$ is the mean distance between crops of cultivar 1 and 2 in year $t$,
- $W_t$ is the weather index affecting the disease in year $t$. 
4.1.3 Fitting functional form to framework

To get a more specific indication of the issues involved, functional forms can be estimated to provide an initial approximation. The probability of "breakdown" is assumed to be proportional to the product of the level of resistance and the amount of inoculum, so that:

\[ B_{1t} = a R_{1t} I_{1t}, \]

where \( a \) is a positive constant, \( R_{1t} \) is the level of resistance of cultivar 1, represented by a value between 0 (immune, highly resistant) and 1 (highly susceptible), and \( I_{1t} \) is the expected amount of inoculum available to that cultivar in that year.

The amount of inoculum from cultivar 1 is assumed to be proportional to the product of the level of resistance, the area sown to that cultivar and the weather index (0 = not favouring the disease at all; 1 = ideal for the disease), and the inoculum available from cultivar 2 is then added:

\[ I_{1t} = b R_{1t} A_{1t} W_t + I_{2t}, \]

where \( b \) is a positive constant.

The function for inoculum from cultivar 2 has to have the following properties:

(a) it must approach zero as the distance becomes very large;
(b) it must be zero if cultivar 2 is immune (\( R_{2t} = 0 \));
(c) it must reduce to the same as the first part of equation (7) (that is, be proportional to the product of its level of resistance and its area) if the two cultivars have the same level of resistance,
(d) the greater the difference in resistance or the smaller the distance between crops (\( D_t \)), the greater the impact of cultivar 2 on cultivar 1;
(e) if the area sown to cultivar 2 is zero (\( A_{2t} = 0 \), \( A_{1t} = A_t \)), the result should be the same as if the two cultivars have the same level of resistance (\( R_{1t} = R_{2t} = R_t \)).

These conditions are met by the following functional form:

\[ I_{2t} = (b R_{2t} A_{2t} W_t)/(R_{2t}/R_{1t})/[1 + c D_t (R_{2t} - R_{1t})]. \]

Thus, from equations (5), (6) and (7):

\[ B_{1t} = a b R_{1t}^2 A_{1t} W_t + (a b R_{2t}^2 A_{2t} W_t )/[1 + c D_t (R_{2t} - R_{1t})]. \]

While there are other possible functional forms that meet the requirements, equation (9) is used as the basis for initial estimations of some of the critical values.
4.2 Some Empirical Examples

4.2.1 Relative probabilities

If we initially assume that (a) half the area is sown to each cultivar \( A_{1t} = A_{2t} = 0.5 \), and (b) the constant \( c \) and the distance \( D_t \) are scaled such that \( cD_t = 1 \), then the relative probabilities of "breakdown" of resistance in cultivar 1 in year \( t \) can be estimated from equation (9) for a range of resistance levels (Table 2). For example, if the resistance levels were 0.4 and 0.6 for cultivars 1 and 2, respectively, then the probability would be 0.12ab\( A_t \).

Table 2: Relative Probabilities\(^a\) of "Breakdown"

for Different Levels of Resistance

<table>
<thead>
<tr>
<th>( R_2 )</th>
<th>0.0</th>
<th>0.2</th>
<th>0.4</th>
<th>0.6</th>
<th>0.8</th>
<th>1.0</th>
</tr>
</thead>
<tbody>
<tr>
<td>( R_1 )</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>0.0</td>
<td>0.00</td>
<td>0.02</td>
<td>0.06</td>
<td>0.11</td>
<td>0.18</td>
<td>0.25</td>
</tr>
<tr>
<td>0.2</td>
<td>0.02</td>
<td>0.04</td>
<td>0.09</td>
<td>0.15</td>
<td>0.22</td>
<td>0.30</td>
</tr>
<tr>
<td>0.4</td>
<td>0.08</td>
<td>0.11</td>
<td>0.16</td>
<td>0.23</td>
<td>0.31</td>
<td>0.39</td>
</tr>
<tr>
<td>0.6</td>
<td>0.18</td>
<td>0.21</td>
<td>0.28</td>
<td>0.36</td>
<td>0.45</td>
<td>0.54</td>
</tr>
<tr>
<td>0.8</td>
<td>0.32</td>
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<td>0.45</td>
<td>0.55</td>
<td>0.64</td>
<td>0.74</td>
</tr>
<tr>
<td>1.0</td>
<td>0.50</td>
<td>0.60</td>
<td>0.70</td>
<td>0.81</td>
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<td>1.00</td>
</tr>
</tbody>
</table>

\( a \): From equation (9), the figures in this table are the value of \( 0.5 \, R_{1t}^2 + 0.5 \, R_{2t}^2/[1+(R_{2t}^2 - R_{1t}^2)] \), that is the coefficient of \( abA_tW_t \).

4.2.2 Estimating size of probabilities

To get an estimate of the size of the constant \( ab \), consider that all the area is sown to cultivar 1 (or both cultivars 1 and 2 have the same level of resistance). Then

(10) \[ B_{1t} = a \, b \, R_{1t}^2 \, A_t \, W_t. \]

If \( R_{1t} = 1 \) (the cultivar is very susceptible to a disease) and \( W_t = 1 \) (weather conditions were ideal for the disease), we expect \( B_{1t} = 1 \). Therefore, we have

(11) \[ a \, b = 1/A_t. \]

If we substitute in equation (9), we get
(12) \[ B_{1t} = R_{1t}^2 W_t \left( \frac{A_{1t}}{A_t} \right) + R_{2t}^2 W_t \left( \frac{A_{2t}}{A_t} \right) \left[ 1 + cD_t \left( R_{2t} - R_{1t} \right) \right] \].

If we now assume that (a) half the total area is sown to each cultivar \( A_{1t} = A_{2t} = 0.5 A_t \), (b) the weather index takes its mean value of 0.5, and (c) the constant \( c \) and the distance \( D_t \) are scaled such that \( cD_t = 1 \), then the absolute values of the probability, \( B_{1t} \), can be estimated (Table 3) by the following form of the previous equation:

(13) \[ B_{1t} = 0.25 R_{1t}^2 + (0.25 R_{2t}^2) \left[ 1 + (R_{2t} - R_{1t}) \right] \].

<table>
<thead>
<tr>
<th>( R_1 )</th>
<th>0.0</th>
<th>0.2</th>
<th>0.4</th>
<th>0.6</th>
<th>0.8</th>
<th>1.0</th>
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<td>0.00</td>
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<td>0.03</td>
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<td>0.09</td>
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<td>0.04</td>
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<tr>
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<td>0.18</td>
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</tr>
<tr>
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<td>0.30</td>
<td>0.35</td>
<td>0.40</td>
<td>0.45</td>
<td>0.50</td>
</tr>
</tbody>
</table>

Table 3: Absolute Probabilities of "Breakdown"
for Different Levels of Resistance

\[ a: \text{For } A_{1t} = A_{2t}, W_t = 0.5, \text{ and } cD_t = 1. \]

In Appendix A, the relationship between the probability of resistance breaking down in a given year and the expected life of resistance is identified. From other data on the observed life of resistances, we expect \( B \) to have a value of approximately 0.12 (see Appendix A). On that basis, solving equation (13) for \( B_{1t} = 0.12 \) and \( R_{1t} = R_{2t} \), the average value we would expect for \( R_{1t} \) is 0.49.

### 4.3 Example of Release of Susceptible Wheat Cultivar

Consider the case of the release of a susceptible wheat cultivar in NSW when all other cultivars have been resistant to, say, stem rust. Assume the following parameter values: \( A_{1t} = 2 \text{ million ha; } R_{1t} = 0.2; W_t = 0.5; R_{2t} = 0.8; A_{2t} = 100,000 \text{ ha (5\%). }\) On the basis of these parameter estimates, the expected value of \( B_{1t} \) without cultivar 2, based on equation (11) with \( W_t = 0.5 \), would be 0.020. The value of \( B_{1t} \) after the release of cultivar 2, based on equation (11) with \( cD_t = 1 \) and \( W_t = 0.5 \), would be 0.029. Therefore, the overall average probability of "breakdown" in the resistant crops increases by 45%. While the probabilities are still low for susceptible crops only reaching 5% of the area, if the area rose to 15%, for example, the
probability would increase by 135% to 0.047, and the expected life of the resistance would be reduced by more than half. Similarly, in the year when the weather favoured the disease ($W_t = 1$), the probability of "breakdown" would have increased substantially.

At a local level, the risks of disease "breakdown" on neighbouring farms are even higher. Table 3 shows the increase in susceptibility of the resistant cultivar if the susceptible crop reached 50% of the area sown. On that basis, the neighbouring resistant crop the same size as the susceptible crop has its probability of "breakdown" increased from 0.02 to 0.11 (Table 3) in an average year, a very significant extra cost for the adjoining farms.

The private costs of growing this cultivar will vary from region to region. However, from Brennan and Murray (1989), the average private costs of a cultivar susceptible to stem rust are approximately 10% for both northern and southern NSW.

5. POLICY OPTIONS FOR CONTROLLING SUSCEPTIBLE CULTIVARS

5.1 Rationale for Government Intervention in Cultivar Choice

Government intervention in the farmers' choice of cultivar can be justified provided there is: (a) some market failure, in that private costs do not provide adequate incentive for farmers to act optimally from the point of view of society, and (b) some cost-effective government action that will modify behaviour to bring about the desired social outcome.

It is apparent that farmers growing susceptible cultivars impose a cost on other farmers who grow resistant cultivars, and that there is market failure in that these externalities are not accounted for in the market. If individual growers were fully penalised for the costs that they impose on other individuals and the aggregate benefits of any such taxes outweighed the costs of calculating and collecting them (NSWWG 1986), the choices made by fully-informed growers will be efficient from the viewpoint of society. However, problems in meeting these conditions may justify a public authority preventing an individual from making a free choice of cultivar. Nevertheless, it is difficult to meet these conditions in a cost-effective way.

There are a limited number of options available to authorities in the face of such externalities and market failure. Two policy options are: (a) regulating cultivar release, and (b) taxes or charges imposed on susceptible cultivars.
5.2 Rationale for Regulating Cultivar Release

5.2.1 Regulating cultivar release

The requirements for a successful scheme of government regulation on varietal control are:

(a) government legislation making it illegal to release or recommend a cultivar not receiving official sanction;
(b) farmers to accept that they should not grow cultivars that are not given official sanction;
(c) full control of all cultivars being put forward for release in a particular environment.

It is unlikely that laws could be enforced to prevent farmers growing cultivars from outside the State should they have inadequate resistance. In the current deregulated environment, it is also unlikely that such restrictions would be adhered to even if the government was persuaded to try to implement them. Johnston et al. (1983) and NSWWG (1986) both argued that, rather than restrict or regulate cultivars released, any government intervention should take the form of a discount/tax on price for susceptible cultivars, reflecting the external costs imposed.

5.2.2 Common disease standards for cultivar release

An important issue in regulating cultivar release is that of common disease standards. The stipulation of minimum disease resistance standards sets a minimum value below which a cultivar is unacceptable irrespective of all other attributes (Doodson 1976). Since epidemics are often the consequence of cultivation of very susceptible cultivars, minimum standards attempt to avoid such types with the aim of reducing the probability of epidemics and the degree of loss.

Minimum disease resistance standards for varietal release and recommendation have been discussed publicly at conferences, and internally in wheat breeding programs in Australia. Minimum disease resistance standards for southern NSW were accepted by Standing Advisory Committee for Wheat in 1987. Particular cultivars were nominated as the minimum disease standard for the major diseases considered in the breeding program. However, setting up such formal standards presented difficulties for other programs (P. Brennan, F. Ellison, R. Hare, R. Young, H. Wallwork, R. Wilson, Personal Communication).

One of the main problems is the likely trade-off between different varietal features. For example, Victorian and South Australian breeders aim for stem rust resistance, but would be likely to release a stem rust susceptible cultivar if it was resistant to cereal cyst nematode (CCN) and high-yielding and of adequate quality. A CCN-resistant cultivar would be more profitable than one with stem rust resistance, given the relative incidence and severity of the two diseases. Thus, cultivars susceptible to
major diseases could still be released, even if they are subjected to minimum disease resistance standards in some States.

Such a release may be advantageous in the short term, but under certain circumstances could be very destructive in the longer term. Consider if stem rust oversummered in South Australia with a predominance of stem rust susceptible cultivars and provided a source of inoculum for the eastern States in a season with favourable conditions for stem rust. This happened in 1990-91 when the \textit{Sr30} attacking pathotype oversummered in SA. Fortunately, the following season did not have favourable weather conditions for stem rust, as there were large areas sown to cultivars susceptible to this pathotype in Victoria and southern NSW.

Another problem is that breeding lines may escape disease in early generations, but show inadequate resistance in more stringent later generation tests. Should they have some other particularly promising feature, such as high yield or quality, there may be pressure to lower disease resistance standards, and indeed there may well be good economic reason for it.

Before cultivars are subjected to desirable or minimum disease standards, breeding programs need to agree on issues such as (a) which diseases are sufficiently important to justify restrictions on cultivar release, (b) whether resistance is available that would result in reduction in yield loss to the disease, and (c) whether there are economic alternatives, such as fungicide that could result in economic disease control.

Given the difficulty in getting breeding programs to agree on an acceptable set of minimum standards, it is unlikely that growers would willingly accept such standards for diseases not important to them. As a result, and because of the difficulty of controlling cultivar release in a deregulated environment, it is not likely that a feasible set of minimum disease standards could be imposed without very high enforcement costs.

5.3 Rationale for Taxes and Charges on Susceptible Cultivars

5.3.1 Form of cultivar tax

An alternative means of government intervention is to impose a tax or charge on susceptible cultivars. The prerequisites for such a tax or charge are: (a) accurate cultivar identification in the market, (b) accurate estimation of the costs imposed by different degrees of susceptibility; and (c) some means of ensuring that those who bear most of the costs of susceptible cultivars would receive most of the benefits from the tax.

In practice, these requirements are not likely to be met in an unregulated grains market. In the fully regulated system applying in Australia before the domestic wheat market was deregulated, it may have been feasible. Indeed, Johnston et al.
(1983) argued that the AWB should impose dockages on disease susceptible cultivars. However, with deregulation and removal of the AWB's acquisition powers, such a tax system would be complex to administer, especially as the basis for the charge would not be related to the grain's value in the market in any of its end uses. For example, feed merchants would be required to collect a tax on a cultivar that did not suffer any quality defect in relation to its use for stockfeed.

The critical question is whether the reduction in aggregate external costs induced by a particular tax or charge would exceed increased costs to farmers and the policy costs of imposing such charges. Research would be needed to define the magnitude of external costs, farmer costs and policy costs with a view to setting charges equal to external costs and to determining the overall costs and benefits of the scheme. However, Johnston et al. (1983) argued that the removal of the AWB monopoly would make it impossible to operate the varietal dockage scheme and hence this proposal. Nevertheless, there may be still a role for regulation of varietal releases in an otherwise deregulated industry.

In view of the difficulties of assessing these benefits and costs it may well be optimal to adopt a mixed dockage/standards approach (see Baumol and Oates 1971, 1975, Weitzman 1974, Roberts and Spence 1976). A decision would be made on the maximum desirable area of susceptible crops in any particular region and then taxes would be raised until current areas were reduced to that standard (Johnston et al. 1983). In the case of stem rust, for example, because of the high damage likely to be done by even small amounts of inoculum, the maximum desirable area is likely to be very small. One particular form of this strategy would be to impose "dockages" for disease control only on those susceptible cultivars for which there was an agronomically very similar resistant substitute and then impose a prohibitive tax on the susceptible cultivar to eliminate use of the cultivar altogether. A similar strategy has been used in the past by the AWB to set dockages for "inferior quality" cultivars.

If a tax were introduced for disease susceptibility, there would be an incentive for those farmers who grew the cultivar to incorrectly identify the cultivar on delivery. Currently no check of varietal identity is made. However, such tests are available (Gore et al. 1990, Wrigley et al. 1989). NSWWG (1986) argued that the aggregate external cost of this mis-specification would probably not be large, since the magnitude of "marketing" quality dockages or implied premiums would be likely to be significantly greater than the "disease" charges. However, this is not likely to be the case as disease charges would be likely to be substantial in some cases.

Some estimate of the size of a tax on susceptible cultivars can be obtained on the basis of some broad assumptions. If we assume that the price of wheat is $100/t on farm, the yield of disease-free wheat is 2.0 t/ha, and the average life of cultivars on farms is 8 years (Brennan and Byerlee 1991), then the expected yields of a farmer with probabilities of a "breakdown" of 0.02 per year and 0.029 per year can be compared (section 4.3 above). Expected average yields over an 8-year life are 1.83 t/ha for the grower of cultivar 1 without cultivar 2, and 1.76 t/ha with cultivar 2. On that basis, the costs for growers of the resistant cultivar would be $7 per ha per
year, or a total of $13.3 million per year for the whole 1.9 million ha. This would require a tax of $133/ha on the susceptible crop to raise this money to be used for compensating the affected growers. Those geographically closest to the susceptible crop would receive the greatest compensation ($30/ha for the next-door neighbour), and those furthest away the least. Thus disease taxes would be substantial, certainly outweighing quality premiums and discounts, and providing strong incentives for mis-specifying cultivars.

5.3.2 Policy costs of dockages/discounts

Even if an appropriate charge can be determined and implemented, there is a cost of gathering information and establishing just what costs or benefits one grower is imposing on others. There would be a cost of administering a charging scheme including the identification of cultivars and preventing growers avoiding or evading any charging scheme. Because these costs are significant, it may not be possible to fully reflect the costs and benefits of an individual grower’s choice of cultivars back to him/her.

Policy costs consist of administrative, information and enforcement costs. Administrative costs for a tax for disease susceptibility would have been negligible under the earlier system, given that some quality payments were already based on cultivar. However, that is not the case in the deregulated system. Information costs such as for research to determine the magnitude of external damage costs for all diseases may well be significant. However a mixed standards/dockage approach would have far lower information costs (NSWWG 1986). Enforcement costs could also be high especially where susceptible cultivars were visually very similar to resistant cultivars in the absence of disease and where there was a significant perceived private cost to some farmers of reducing their plantings of the susceptible cultivar.

5.4 Implications for Policy

Therefore, while it seems that there are considerable costs imposed by growers who grow susceptible cultivars, especially in the vicinity of other formerly resistant crops, there are no easy methods of preventing growers from such a course of action. It seems that the costs of imposing and policing such a policy would be prohibitive, either in terms of administrative and enforcement costs or in terms of restrictions on the freedom and behaviour of farmers.

Therefore, it seems that the only role for government intervention in this debate is that of information. The importance of the costs imposed on others needs to be emphasised at all times, as well as the private costs and risks that those growing susceptible cultivars, even if they are higher-yielding in disease-free situations, need to be emphasised.

One example of the importance of the information role of government is the
Queensland Department of Primary Industries (QDPI) publicity campaign on the withdrawal of the cultivar Cook. The use of the cultivar Oxley continued in central Queensland after a pathotype virulent on it had been recorded, thus providing a means for the progressive accumulation of virulences and for the evolution of the Cook pathotype. This new strain with virulence on Sr36 in Cook was identified late in 1984. The QDPI actively sought the prompt withdrawal of Cook, even though seed for the new season had already been harvested and set aside (P. Brennan, Personal Communication). The percentage of the area sown to Cook fell from 27.6% in 1984 to 14.7% in 1985 and to zero by 1987. The prompt action of QDPI in providing and promoting that information certainly minimised the losses from the new pathotype.

6. CONCLUSIONS

It is apparent from the above analysis that there is a strong case for saying that there is market failure in this area. The social costs of a grower's actions are not reflected in his private costs. Policy options to overcome this market failure have been identified. However, following the deregulation that has taken place in the wheat industry, the costs of the various policy options available are likely to have increased sharply and to be extremely high. As a consequence, there appears to be no cost-effective policy that would bring about the socially desirable revision of growers' behaviour. The only remaining option is a widespread publicity campaign. It is likely to have only a limited impact, since it will require farmers to pay a private cost for the public benefit. Therefore, the additional costs of disease losses by growers that are caused by others growing susceptible cultivars, and the costs of that campaign, are costs of the policy of deregulation. Such policy costs need to be identified and examined more closely before further deregulation takes place.
REFERENCES


National Academy of Sciences (1972), *Genetic Vulnerability of Major Crops*. Washington, DC.


If the probability of the genetic resistance of a cultivar breaking down in any one year be $B_i$ (0 $< B_i < 1$). Then the probability of resistance of the gene surviving the year is $(1 - B_i)$. If we assume initially that the probability of breaking down is independent from one year to another and is a constant, $B$, then the probability of resistance surviving for $n$ years, $S_n$, is:

(A.1) $S_n = (1 - B)^n$.

The number of years for which there is a 50% probability that the resistance of a variety will survive, $N$, can be calculated, by solving equation (A.1) for $n = N$ and $S_n = 0.50$. $N$ is calculated by:

(A.2) $N = \log(0.50)/\log(1 - B)$.

The estimated values of $N$ are shown in Table A.1 for different values of $q$.

<table>
<thead>
<tr>
<th>Probability of resistance breaking down in given year</th>
<th>50% probability of surviving for:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(years)</td>
</tr>
<tr>
<td>0.1</td>
<td>6.6</td>
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<tr>
<td>0.5</td>
<td>1.0</td>
</tr>
</tbody>
</table>

Alternatively, we can use information on the observed life of resistance to infer an average value of $B$, as equation (A.1) can be rewritten as:

(A.3) $B = 1 - S_n^{(1/n)}$.

From Kilpatrick (1975), the frequency of wheat rust race changes was found to vary from 1 to 15 years, with a weighted average of 5.2 years. For $n = 5.2$ and $S_n = 0.5$, we have $B = 0.12$. 